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Downregulation of CK2 induces apoptosis in cancer cells--a potential approach to cancer therapy.

Wang G, Unger G, Ahmad KA, Slaton JW, Ahmed K.

Cellular and Molecular Biochemistry Research Laboratory (151), Minneapolis Veterans Affairs Medical Center, Minneapolis, MN 55417, USA.

We have previously documented that naked antisense CK2alpha ODN can potently induce apoptosis in cancer cells in culture and in mouse xenograft human prostate cancer. The effects of the antisense CK2alpha are related to downregulation of CK2alpha message and rapid loss of the CK2 from the nuclear compartment. Here we demonstrate that downregulation of CK2 elicited by diverse methods leads to inhibition of cell growth and induction of apoptosis. The various approaches to downregulation of CK2 employed were transfection with kinase-inactive plasmid, use of CK2alpha siRNA, use of inhibitors of CK2 activity, and use of antisense CK2alpha ODN packaged in sub-50 nm nanocapsules made from tenascin. In all cases, the downregulation of CK2 is associated with loss in cell survival. We have also described preliminary observations on an approach to targeting CK2 in cancer cells. For this, sub-50 nm tenascin-based nanocapsules bearing the antisense CK2alpha ODN were employed to test that the antisense is delivered to the cancer cells in vivo. The results provide the first preliminary evidence that such an approach may be feasible for targeting CK2 in cancer cells. Together, our results suggest that CK2 is potentially a highly plausible target for cancer therapy.

PMID: 16342410 [PubMed - indexed for MEDLINE]

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